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Surface molecules involved in the activation and regulation of T or natural killer lymphocytes in humans.

Moretta A, Ciccone E, Pantaleo G, Tambussi G, Bottino C, Melioli G, Mingari MC, Moretta L.

Istituto di Istologia ed Embriologia, Universita'di Genova, Italy.

The surface molecules that mediate activation of different subsets of T or NK cells have been reviewed. A suitable approach to the study of different lymphocyte activation pathways is provided by mAbs specific for these molecules. MAbs directed to the CD3 surface molecules mediate a polyclonal T-cell activation, whereas mAbs to "clonotypic" structures of TCR only trigger cells bearing the corresponding clonotypic determinant (thus mimicking the effect of antigen/MHC). MAbs directed to appropriate epitopes of CD2 molecules or to CD28 molecules mediate polyclonal T-cell activation, leading to triggering of the functional program of the cell (i.e. proliferation, lymphokine production or activation of the cytolytic machinery). Interaction of specific mAbs with CD3/TCR molecules leads to surface modulation of these molecules which lasts for 48-72 h. During this interval the cell is refractory to any further activation stimulus. No such refractoriness occurs following mAb-induced modulation of CD2 or CD28 surface molecules. The mechanisms by which CD3/TCR modulation results in the inactivation of T-cell function appears to involve the early metabolic steps of T-cell activation, as neither Ca++ mobilization nor IP3 formation could be further induced by any stimulus. The surface molecules and mechanisms involved in the activation of TCR gamma/delta cells are similar to those of TCR alpha/beta + cells. TCR gamma/delta molecules are heterogeneous in size and charge mobility. MAbs directed to one or another form of TCR gamma/delta trigger the functional program of the cell (primarily cytolytic function). However, a receptor form composed of a heavy form (55 kD) of the gamma chain appears to be relatively inefficient in signal transduction upon binding with anti-TCR mAbs. Evidence has also been provided that TCR gamma/delta + cells are capable of (allo)antigen responses and that polymorphic determinants of class I can be recognized (specific lysis of P815 cells transfected with HLA-A24 allele). Although the mechanisms and the surface receptor molecules involved in (CD3-, CD16+) NK cell activation are still poorly understood, several surface molecules have been identified that mediate NK-cell triggering. These include CD2 and CD16 and the novel GL183 molecule which is selectively expressed by a fraction of NK cells and thus identifies a well-defined NK subsets. Under appropriate conditions, mAbs to CD16 or GL183 mediate an inhibitory effect on the NK cell activation. These data suggest that also NK cells are characterized by surface molecules capable of initiating distinct pathways of cell activation and that, similarly to T lymphocytes, mechanisms exist which regulate NK cell function.